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NEWS	4	APR 04	STN AnaVist $500 visualization usage credit offered
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NEWS	6	MAY 11	KOREAPAT updates resume
NEWS	7	MAY 19	Derwent World Patents Index to be reloaded and enhanced
NEWS	8	MAY 30	IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2
NEWS	9	MAY 30	The F-Term thesaurus is now available in CA/CAplus
NEWS	10	JUN 02	The first reclassification of IPC codes now complete in INPADOC
NEWS	11	JUN 26	TULSA/TULSA2 reloaded and enhanced with new search and and display fields
NEWS	12	JUN 28	Price changes in full-text patent databases EPFULL and PCTFULL
NEWS	13	JUl 11	CHEMSAFE reloaded and enhanced
NEWS	14	JUl 14	FSTA enhanced with Japanese patents
NEWS	15	JUl 19	Coverage of Research Disclosure reinstated in DWPI
NEWS	16	AUG 09	INSPEC enhanced with 1898-1968 archive

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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=> FIL MEDLINE BIOSIS CA EMBASE SCISEARCH

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.42	0.42

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=> s crem or (camp response element modulator?)
L1         2085 CREM OR (CAMP RESPONSE ELEMENT MODULATOR?)

=> s antisense or anti-sense or (complem? (2n) (oligonucl? or nucle?))
L2       166443 ANTISENSE OR ANTI-SENSE OR (COMPLEM? (2N) (OLIGONUCL? OR NUCLE?)
                )

=> s l1 and l2
L3           95 L1 AND L2

=> s lupus or sle
L4       205041 LUPUS OR SLE

=> s l2 and (l3 or (t cell or leukocyt?))
<------User Break------>

=> s l3 and (l4 or (t cell or leukocyt?))
L5           39 L3 AND (L4 OR (T CELL OR LEUKOCYT?))

=> s l5 and py<=2003
   1 FILES SEARCHED...
L6           23 L5 AND PY<=2003

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7             7 DUP REM L6 (16 DUPLICATES REMOVED)

=> d l7 ibib abs 1-7

L7   ANSWER 1 OF 7  CA  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         139:31807  CA
TITLE:                    Use of HLA-DRB1*1502 gene as marker for sarcoidosis
                          and methods for diagnosis and therapy
INVENTOR(S):              Chibout, Salah-Dine; Grenet, Olivier; Kehren, Jeanne;
                          Staedtler, Frank
PATENT ASSIGNEE(S):       Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE:                   PCT Int. Appl., 65 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     --------------       ----   --------    --------------------    --------
     WO 2003046578         A2    20030605    WO 2002-EP13448         20021128 <--
     WO 2003046578         A3    20040325
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
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HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LT, LU,
LV, MA, MD, MK, MN, MX, NO, NZ, OM, PH, PL, PT, RO, RU, SE, SG,
SI, SK, TJ, TM, TN, TR, TT, UA, US, UZ, VC, VN, YU, ZA, ZW
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE,
DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR

AU 2002364277	A1	20030610	AU 2002-364277		20021128 <--
EP 1454145	A2	20040908	EP 2002-799048		20021128
JP 2005510251	T2	20050421	JP 2003-547966		20021128
US 2005032062	A1	20050210	US 2004-497349		20041001
PRIORITY APPLN. INFO.:			US 2001-334264P	P	20011129
			WO 2002-EP13448	W	20021128

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK

AB This invention identifies genes and the mRNA and polypeptide expression products of these genes which can be used as biomarkers to provide diagnostic and prognostic information in patients with sarcoidosis. These biomarkers can also be used to monitor the severity and progression of sarcoidosis and to identify drugs useful in treating the disease. In particular it relates to expression of HLA-DRB1*1502 gene for histocompatibility antigen MHC class II and its association with sarcoidosis type I, II and III.

L7 ANSWER 2 OF 7 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER:	2003113635 MEDLINE
DOCUMENT NUMBER:	PubMed ID: 12626549
TITLE:	The cyclic adenosine 5'-monophosphate response element modulator suppresses IL-2 production in stimulated **T cells** by a chromatin-dependent mechanism.
AUTHOR:	Tenbrock Klaus; Juang Yuang-Taung; Tolnay Mate; Tsokos George C
CORPORATE SOURCE:	Department of Cellular Injury, Walter Reed Army Institute of Research, Silver Spring, MD 20910, USA.
CONTRACT NUMBER:	R01-AI49954 (NIAID)
SOURCE:	Journal of immunology (Baltimore, Md. : 1950), **(2003 Mar 15)** Vol. 170, No. 6, pp. 2971-6. Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY:	United States
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:	200306
ENTRY DATE:	Entered STN: 11 Mar 2003 Last Updated on STN: 26 Jun 2003 Entered Medline: 25 Jun 2003

AB The production of IL-2 is tightly controlled by several transcription factors that bind to the IL-2 promoter. The **cAMP response element modulator** (**CREM**) is known to form complexes with CREB and bind to the -180 site of the IL-2 promoter in anergic and in systemic **lupus** erythematosus **T cells**. In this study we show that **CREM** is transcriptionally induced in **T cells** following stimulation through CD3 and CD28, binds to the IL-2 promoter in vivo, and suppresses IL-2 production. Transfection of an **antisense CREM** plasmid into **T cells** blocked the expression and binding of **CREM** to the IL-2 promoter and the decrease of IL-2 production, which follows the early increase after **T cell** stimulation with CD3 and CD28. In addition, as assessed by chromatin immunoprecipitation experiments, **antisense CREM** prevented the binding of protein 300 and cAMP response element binding protein and promoted the acetylation of histones. **Antisense CREM** also enhanced the accessibility of the IL-2 promoter to endonucleases and prevented the condensation of chromatin in vivo. Our data suggest that upon **T cell**

activation, **CREM** gradually replaces phosphorylated CREB at the -180 site of the IL-2 promoter. **CREM**, in turn, binds protein 300 and cAMP response element binding protein, but **CREM** is unable to activate its histone acetyltransferase activity, which results in condensation of chromatin and down-regulation of IL-2 production.

L7 ANSWER 3 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 2
ACCESSION NUMBER: 2003:292284 BIOSIS
DOCUMENT NUMBER: PREV200300292284
TITLE: Rewiring the **T-cell**: Signaling defects and novel prospects for the treatment of **SLE**.
AUTHOR(S): Tsokos, George C. [Reprint Author]; Nambiar, Madhusoodana P.; Tenbrock, Klaus; Juang, Yuang-Taung
CORPORATE SOURCE: Department of Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD, 20814, USA
gtsokos@usa.net
SOURCE: Trends in Immunology, (**May 2003**) Vol. 24, No. 5, pp. 259-263. print.
ISSN: 1471-4906 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 Jun 2003
Last Updated on STN: 25 Jun 2003

AB Activation of **T cells** from patients with systemic **lupus** erythematosus (**SLE**) leads to increased signaling responses, detected by increased calcium and protein tyrosine phosphorylation patterns. This overexcitability occurs in spite of decreased levels of **T-cell** receptor zeta chain. The replacement of the zeta chain by the Fc receptor (FcR) gamma chain and the formation of signaling molecule aggregates on the surface of **T cells** are considered to be responsible for the observed signaling phenotype. Decreased production of the zeta-chain promoter binding form of the transcription factor Elf-1 is responsible for the decreased transcription of the zeta chain gene. In addition, transcription of the interleukin-2 (IL-2) gene is decreased because of the presence of the transcriptional repressor cyclic adenine mono-phosphate (**cAMP**) **response element modulator**. Replenishment of the zeta chain and elimination of the repressor by **antisense** approaches leads to increased expression of IL-2, suggesting that gene therapy approaches might represent tangible modalities in the treatment of human **SLE**.

L7 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2002613466 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12370343
TITLE: **Antisense** cyclic adenosine 5'-monophosphate response element modulator up-regulates IL-2 in **T cells** from patients with systemic **lupus** erythematosus.
AUTHOR: Tenbrock Klaus; Juang Yuang-Taung; Gourley Mark F; Nambiar Madhusoodana P; Tsokos George C
CORPORATE SOURCE: Department of Cellular Injury, Walter Reed Army Institute of Research, 503 Robert Grant Avenue, Silver Spring, MD 20910, USA.
CONTRACT NUMBER: R01 AI 49954 (NIAID)
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (**2002 Oct 15**) Vol. 169, No. 8, pp. 4147-52.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200211

ENTRY DATE: Entered STN: 10 Oct 2002
Last Updated on STN: 14 Dec 2002
Entered Medline: 27 Nov 2002

AB The **cAMP response element modulator** (**CREM**) has been shown to bind specifically to the -180 site of the IL-2 promoter in vitro. **CREM** protein is increased in **T cells** of patients with systemic **lupus** erythematosus (**SLE**), and it has been considered responsible for the decreased production of IL-2. In this work we show that transcriptional up-regulation is responsible for the increased **CREM** protein levels and that **CREM** binds to the IL-2 promoter in live **SLE T cells**. Suppression of the expression of **CREM** mRNA and protein by an **antisense CREM** plasmid, which was force expressed in **SLE T cells** by electroporation, resulted in decreased **CREM** protein binding to the IL-2 promoter and increased expression of IL-2 mRNA and protein. Our data demonstrate that **antisense** constructs can be used to effectively eliminate the expression of a transcriptional repressor. This approach can be used therapeutically in conditions where increased production of IL-2 is desired.

L7 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
DUPLICATE 4

ACCESSION NUMBER: 2002:370484 BIOSIS
DOCUMENT NUMBER: PREV200200370484
TITLE: **Anti-sense cAMP response element modulator** (**CREM**) upregulates interleukin 2 mRNA in normal and **SLE T cells**.
AUTHOR(S): Tenbrock, Klaus [Reprint author]; Juang, Yunag-Taung [Reprint author]; Gourley, Mark F.; Tsokos, George C. [Reprint author]
CORPORATE SOURCE: Cell Injury, Walter Reed Army Institute of Research, 503 Robert Grant Avenue, Silver Spring, MD, 20910, USA
SOURCE: FASEB Journal, (**March 22, 2002**) Vol. 16, No. 5, pp. A1044. print.
Meeting Info.: Annual Meeting of Professional Research Scientists on Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jul 2002
Last Updated on STN: 3 Jul 2002

AB The **cAMP response element modulator** (**CREM**) has been previously shown to bind specifically to the 180-site of the IL-2 promoter. **CREM** is increased in patients with systemic **lupus** erythematosus (**SLE**), who have decreased levels of IL2. **T cells** of **SLE** patients and healthy controls were transfected by electroporation with an IL2 promoter-luciferase construct and either a sense **CREM** (S-**CREM**) or an **anti-sense CREM** (AS-**CREM**) or an empty vector plasmid. Compared to the empty vector plasmid, AS-**CREM** increased the luciferase activity while S-**CREM** decreased the luciferase activity of the IL2-promoter construct. In accordance with these results IL-2 mRNA was increased after transfection with the AS-**CREM** plasmid and decreased after transfection with the S-**CREM** plasmid compared to the empty vector. **CREM** protein was increased in western blots after transfection with S-**CREM** and decreased after transfection with AS-**CREM**. HSP 70 mRNA and protein were not affected. In conclusion transfection with either S-**CREM** or AS-**CREM**

upregulates or downregulates **CREM**, respectively, in a specific manner in normal and **SLE T cells**. We propose that **CREM** can serve as potential target for gene therapy with **anti-sense** construct in **SLE** patients with reduced IL2-production.

L7 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:578961 BIOSIS
DOCUMENT NUMBER: PREV200200578961
TITLE: **Anti-sense cAMP response element modulator** (**CREM**) upregulates interleukin 2 mRNA in normal and in **SLE T cells**.
AUTHOR(S): Tenbrock, Klaus [Reprint author]; Juang, Yuang-Taung [Reprint author]; Gourley, Mark F.; Tsokos, George C. [Reprint author]
CORPORATE SOURCE: Cell Injury, Walter Reed Army Institute of Research, Silver Spring, MD, USA
SOURCE: Journal of Investigative Medicine, (**March, 2002**) Vol. 50, No. 2, pp. 178A. print.
Meeting Info.: 2002 Clinical Research Meeting. Baltimore, MD, USA. April 10-13, 2002. American Federation for Medical Research; Association for Patient-Oriented Research.
ISSN: 1081-5589.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002

L7 ANSWER 7 OF 7 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2000450791 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11002260
TITLE: Repression of tax-mediated human t-lymphotropic virus type 1 transcription by inducible cAMP early repressor (ICER) protein in peripheral blood mononuclear cells.
AUTHOR: Newbound G C; O'Rourke J P; Collins N D; Andrews J M; DeWille J; Lairmore M D
CORPORATE SOURCE: Center for Retrovirus Research and Department of Veterinary Biosciences, Ohio State University, Columbus, Ohio, USA.
CONTRACT NUMBER: AI01474 (NIAID)
CA55185 (NCI)
P30 CA 1058 (NCI)
SOURCE: Journal of medical virology, (**2000 Oct**) Vol. 62, No. 2, pp. 286-92.
Journal code: 7705876. ISSN: 0146-6615.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 1 Nov 2000

AB Human T-lymphotropic virus type 1 (HTLV-1) infection causes adult **T-cell** leukemia and is characterized by long periods of clinical latency with low levels of viral production. Transcription of HTLV-1 is controlled through sequences in the promoter and enhancer regions of the long terminal repeat of the integrated provirus. Important among these sequences are three 21 bp imperfect repeats responsive to the viral oncogenic protein Tax (TRE). Members of the CREB/ATF-1/**CREM** family of transcription factors bind to TRE-1 and are critical for HTLV-1 transcription. Other less studied family members include the inducible cAMP early repressor (ICER) proteins. ICER proteins lack phosphorylation

and activation domains and are potent inhibitors of transcription. The ability of ICER to bind TRE-1 and its effects on HTLV-1 Tax mediated transcription have not been studied in the natural cell targets of the virus, peripheral blood mononuclear cells (PBMC). We show that ICER mRNA levels are low in quiescent PBMC, but rise and remain elevated for up to 18 hr after mitogenic stimulation of these cells. Electrophoretic mobility shift assays using recombinant Tax and ICER demonstrate that ICER binds TRE-1 and that binding is increased in the presence of Tax. Furthermore, over expression of ICER IIgamma suppressed Tax-mediated transcription whereas an **anti-sense** ICER II plasmid designed to block endogenous ICER enhanced Tax-mediated transcription in activated PBMC. Together our data indicate that ICER inhibits Tax-mediated transcription in activated PBMC and suggest a role for ICER in maintenance of HTLV-1 persistence.